With the author's regards

[*From Biometrika, Vol. VI. Part 1. March*, 1908.]

Not to be removed

Extract ~~Kellie~~ of Letter from Dr L. to Dr Kellie.

Edin. Med & Surg. Journ. 1808. Vol 4. p 252

She told us the following pretty story concerning the origin of this strange defect, which may deserve a place among the legends of the nursery, if not in your Journal.

A clergyman, who had taken great delight in the cultivation of fruit trees, had long suffered keen disappointment, in consequence of the barrenness of a favourite tree, which he expected would yield an uncommonly fine kind of fruit. At last some appeared, they were approaching to that state of maturity which would enable him to determine the all-important point; when, notwithstanding a strict charge which he had given to his gardener, to allow no person to enter the garden, the apples disappeared. Enraged, he taxed the gardener with the theft which he strongly denied. In reply to a charge of disobedience of orders, he affirmed that he could not have supposed that these were meant to exclude his mistress. The parson's lady was then in a state of pregnancy. Her husband inquired softly, whether her longing had tempted her, like our original mother, to take the strictly forbidden fruit? She said no. The gardener was now accused with the utmost violence; and the cool assertion of his innocence only contributed to the ~~the~~ transformation of the divine

into a demon. In that state he pushed into his wife's presence, & with dreadful rashness wished, that if she was guilty, the child which she was then with might be born without fingers! — Poor woman! she had indeed taken the print; & thus became the grand-progenitrix of a fingerless race, unto (even now) the tenth generation.

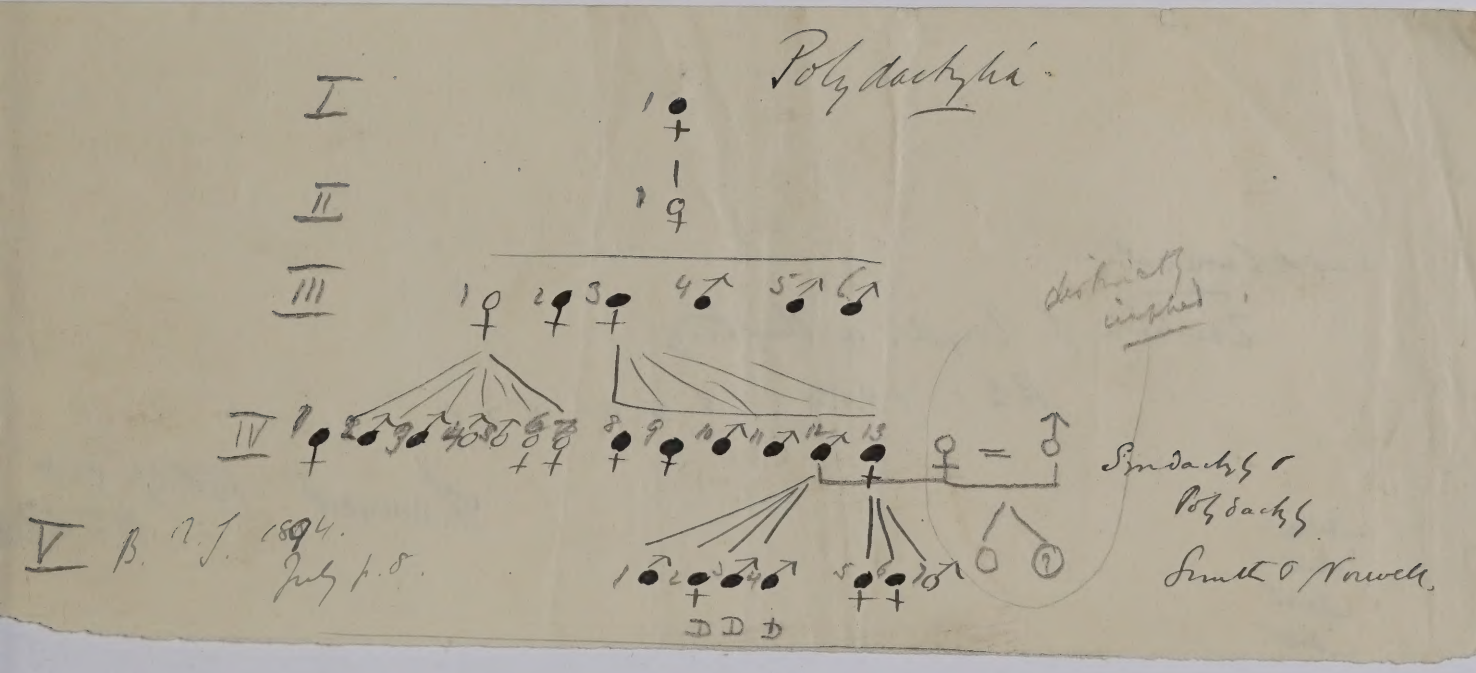
Hereditär Syfilis fort

Schwalbe.
Münch. med. Woch. 13. Mai. 06.
S. 493

Fest Epileptical, handed. digits 4 & 5
Grandchildren.

Parents were unaffected
a ~~younger~~ brother who
died earlier had some
or similar deformity.

Further cases (hereditary) by
Schwyzinberg Virchows Archiv
Bd 193. 1908. S. 1.


Polydactyli
I
II
III
IV
V
distinctly implied
Syndactyli or
Polydactyli
Smith & Norwell

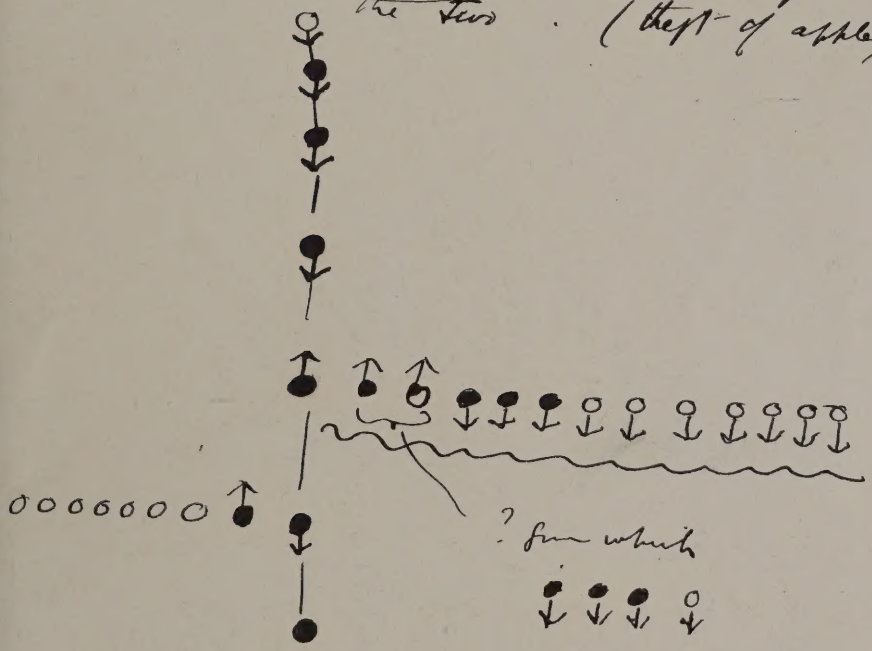
Hasselwander.

Zeitschr f. Anth. u Ethnol.
Bd 1. 1903. S. 510-526

Lancet. 1884. Oct.
25. p. 7

~~22~~
~~43~~
~~46~~
~~50~~

Hackmeles. James borough family
Keelie's family was Uxbridge.
the same story is told of
~~the two~~. (theft of apple).



Remarks of Wilson on Polydactylism in man.

"Kaufertins estimates that polydactylism does not occur so frequently as in one person in 20,000; if that estimate be taken for postaxial additions (little finger side) only, one may well assume that preaxial additions (thumb side), which are much rarer, are not so frequent as in one person in 100,000; ---. So it is not likely that my five Scotch cases (2 of which are here twins) show two unconnected abnormalities;"

Wilson: p. 443.

"In the great majority of cases of hereditary polydactylism, with which I am acquainted, the abnormality is very constant in position though not in degree." "It is not even a rare thing to find the abnormality passing from the outer side of the hand to the outer side of the foot & vice versa; but it is a rare fact --- the passing of the abnormality from the one side of a limb to the other. Wilson p. 442.

Many references given. See also Teratological Reports etc

Correct.

Refers to parallel work of TL & Gambleton.
Enclosing 6pp. TL MSS, including anecdote of apples.

Polydactylism

1. Atavistic in origin (Bardeleben)
2. Intrauterine injuries (Ahlfeld, Zander)
3. Abnormality from variation of germ plasm (Förster, Weismann)

} Intra uterine

"Dichotomy" according to Sutton ("Evolution & disease", London, 1891.)

✓ Bardeleben. Verhdl. d. Anat. Ges., Jahrg. 8, 1894.

Check. { Ahlfeld. "Missbildungen des Menschen," Leipzig, 1880.
Zander. Virch. Archiv., Bd. 125, 1891.

rest correct.

Editorial Note.

The importance of certain considerations being borne in mind with regard to pedigree work is well illustrated by Dr Lewis' note. On looking at Hasselwander's pedigree I personally had considered Dr Lewis' statement correct, not because of the misprint which might attribute some of the children of Fanny to Victoria, a normal individual (for the numbering of the offspring showed that there was a misprint of some sort), but because the writer states directly that the deformed Josepha K. was the child of normal parents.

Now it is at this point that a very important consideration comes in. The true Mendelian needs the theory of mutations to supplement his position, and other workers, among whom, I think, I may include Dr Lewis, would say that the deformity started in a sport. Now an experience covering many hundreds of human pedigrees leads me to the conclusion, that the appearance of deformity in the offspring of normal parents with nothing further known of the ancestry is too often only another way of stating that we have not been able to trace fully the ancestral and collateral lines. In very many cases where we can go back through a normal parentage, I find directly or collaterally the supposed mutation or sport recurring, and we have either to assert (i) that there exists a general tendency to sporting in the stock, or (ii) that the deformity is latent and can be carried through normal individuals. It is needless to say that (ii) appears to me a more scientific assumption. To stop short at a normal pair with the statement 'sport' or 'mutation' is logically incomplete, if we wish to demonstrate that the deformity is handed down only through deformed individuals. We must show for a generation or two in the direct and collateral lines an entire absence of the abnormality.

It is a very general experience that these abnormal cases belong to the lower classes of the population and that in going back in the pedigree we are invariably checked at but a few generations by ignorance as to blood-relationships. Under such circumstances we must either wind up with an abnormal individual of unknown parentage or with normal individuals as in Dr Hasselwander's or Dr Lewis' cases. My first consideration is that we have not the evidence necessary to speak of the latter as determining a 'sport.'

My second important consideration is this: Is not the time ripe for the collection and publication of pedigrees bearing upon normal and abnormal characters in man? There is an immense amount of published material scattered through a wide journalistic area. There is a growing tendency for medical men to note more and more completely family histories*. A collection of published and unpublished material would be of first class value to students of heredity. The primary conditions for such a collection are: (*a*) that it shall contain the material only; no discussion of hereditary theories shall form part of its plan,—this ought to allow of all schools contributing; (*b*) that there shall be no selection of special pedigrees for publication, except on the score of their completeness and the peculiar interest of their characteristics; (*c*) that each contributor should be wholly responsible for the accuracy of his facts and genealogies which should be published under his own name. The duty of the Editor should only be to standardise the material received. With a view to providing a collection of this kind, the Galton Laboratory of National Eugenics proposes to issue in parts a *Thesaurus of Human Inheritance*, each part will contain 20 to 50 pedigrees arranged on 10 lithographed plates. The individual plates will be devoted to a special characteristic or abnormality. Each pedigree will be accompanied by a brief account of the family and its members, and when needful by an illustration of the characteristic. The *Thesaurus* will cover published and unpublished material, and there will be an entire absence of any theoretical discussions. Full reference will be given to the source of the pedigree and to treatises where similar cases are discussed. The Laboratory has already received promises of help from members of the medical profession, and from officials in asylums, sanitoria and hospitals for special diseases. Is it asking too much to beg our Mendelian friends to give us cooperation in this matter, or at any rate to decide on the basis of the first number whether they cannot do so? The need for such a repertorium must be as manifest to them, as to any other students of heredity, and it would add much to the completeness of our scheme if they did not stand aloof from it. The preparation of material for Part I is well advanced and it is hoped to issue it in October.

* Several hundreds of such pedigrees have already reached the Francis Galton Laboratory for National Eugenics.

Editorial Note.

The importance of certain considerations being borne in mind with regard to pedigree work is well illustrated by the Lewis' case. The Doctor of Massachusetts's pedigree, I personally had considered by family statement correct, was an area of the imprint which is not attributable to the children of Sunny to Victoria, a [illegible] individual (for the numbering of the children) [illegible] was a imprint of [illegible] well, but because the within states directly that the [illegible] K. was the child of normal parents.

Now is it at this point that a very important consideration comes in. The true Mendelian needs the theory of mutations to supplement his position, and other workers among whom I [illegible] I may include Dr Lewis, would say that the deformity started in a sport. Now an experience covering many hundreds of human pedigrees leads me to the conclusion, that the appearance of deformity in the offspring of normal parents with nothing to their known of the ancestry is too often only another way of stating that we have not been able to trace fully the ancestral and collateral lines. In very many cases where we can go back through a normal ancestry, I find fibroid or collateral like a [illegible] mutation or sport reporting, and we have [illegible] (i) that there exists a general tendency to sporting in the stock, or (ii) that the [illegible] in taking up can be carried through normal individuals. It is needless to say that (ii) [illegible] a [illegible] assumption. To stop short at a normal pair with the statement 'Type [illegible] Mendelian' is logically incomplete, if we wish to demonstrate that the deformity is handed down only through deformed individuals. We must show for a generation or two [illegible] and collateral lines an entire absence of the abnormality.

It is a very general opinion that these abnormal cases belong to the lower classes of the population and that in going back in the pedigree we are invariably checked at last a few generations by ignorance or to blood relationships. Under such circumstances we must either wind up with an abnormal individual of unknown parentage or with normal individuals as in Dr Hassenstein's case in the Lewis' cases. My first consideration is that we have not the evidence necessary to [illegible]

My second consideration is this: Is not the time ripe for the collection and publication of pedigrees showing upon normal and abnormal characters in man? There is an immense amount of published material available, if only a wide journalistic area. There is growing demand for material [illegible] to make more and more completely family biographies. A collection of published and unpublished material would be of great educational value. The primary qualifications for such a collection are: (a) that it shall contain the matter only; [illegible] [illegible] shall form part of the plan—this might to allow of all schools [illegible]; (b) that there shall be no selection of special pedigrees for publication, except on the ground of their complexity and the possible interest of their characteristics; (c) that such [illegible] shall be wholly responsible for the accuracy of his [illegible] and genealogies which should be published under his own name. The duty of the Editor should only be to standardize the [illegible] material [illegible]. With a view to providing a collection of this kind, the Galton Laboratory of National Eugenics is prepared to issue in parts a Museum of Human Inheritance, each part will contain 10 to 20 pedigrees arranged on 10 uniformed plates. The individual plates will be [illegible] in a special manuscript file or addressograph. Each pedigree will be accompanied by a brief summary of the family and its members and when needful by an illustration of the characteristic. The Museum will cover published and unpublished material, and there will be an [illegible] of any theoretical discussion. Full references will be given to the source of the pedigree and to traditions where direct evidence cannot be discovered. The Laboratory has already received pedigrees or help from members of the medical profession, and from officials in asylums and hospitals for mental diseases. It is asking too much to beg our Mendelian friends to give us [illegible] [illegible] to decide on the basis of the most [illegible] [illegible] [illegible] The need for such a museum must be as manifest to them, as to any other students of heredity, and it would add much to the completeness of our collections if they did not stand aloof from it. The preparation of material for Part I is well advanced and it is hoped to issue it in October.

* Several hundreds of such pedigrees have already reached the Francis Galton Laboratory for National Eugenics.

[*From Biometrika, Vol. VI. No. 1. March,* 1908.]

ON INHERITANCE OF THE DEFORMITY KNOWN AS SPLIT-FOOT OR LOBSTER-CLAW.

By KARL PEARSON, F.R.S.

(1) My attention was drawn at the beginning of 1907 to the existence of a family in which "split-foot" or "lobster-claw" was an hereditary deformity. I was unaware at the time that Dr Thomas Lewis and Mr Dennis Embleton were at work on a much more elaborate study of the same subject based on quite different material, and when I did know I did not hand over my material to them, as I ought properly to have done, because I wanted to ascertain independently to what extent simple Mendelism really applied to an obviously inherited and fairly simple human deformity. I wanted to convince myself that Mendelism does or does not apply to such cases, by handling the material myself and investigating by all the means at my disposal the authenticity of the records. At the same time my readers will suffer from having nothing like the completeness of detail in my case which may be found in the earlier paper of this number.

The members of the family are scattered through an agricultural district some distance from London, and I could only afford the cost of bringing three members up to London for radiography. In this matter I must mention my deep indebtedness to Miss C. O. Stevens not only for much aid in following up the family history from the clue she first reported to me, but also for the arduous task of piloting the strangers through the sights of the Metropolis. To Dr Mackenzie Davidson I owe the further big debt that, in the interests of science, he gave up a large portion of his valuable time to preparing by stereoscopic radiography no fewer than 22 negatives of the feet and hands of these three individuals.

Only a few of these radiograms are reproduced in this paper, but the variety of positions taken, as well as the stereoscopic nature of the pictures, has enabled my colleague, Professor Thane, to give a very complete account of the hands and feet of these three individuals. The ever ready aid given by Professor Thane to biometric inquiry would be very inadequately expressed by a few lines of thanks in a single biometric memoir.

It would indeed be impossible from Professor Thane's account of these three cases to predict the precise nature of the deformity—the presence or absence of bones—in other members of the family who have not been radiographed. No

great faith is to be put in the family record of the number of perfect fingers or toes possessed by dead or living members. The list given on pp. 74, 75 must only be trusted for such obvious and gross variations in the nature of the deformity as supernumerary digits or syndactylised fingers. The importance of the accurate description of each individual member, however desirable, is less, after the work of Messrs Lewis and Embleton; their memoir fairly indicates the range of types to be expected. These types are largely represented in the members of my family who have been more fully examined; there are certain additional types also. It is clear that what they and I are dealing with is an hereditary deformity of the hands and feet, with a definite, if remarkably wide, field of variation.

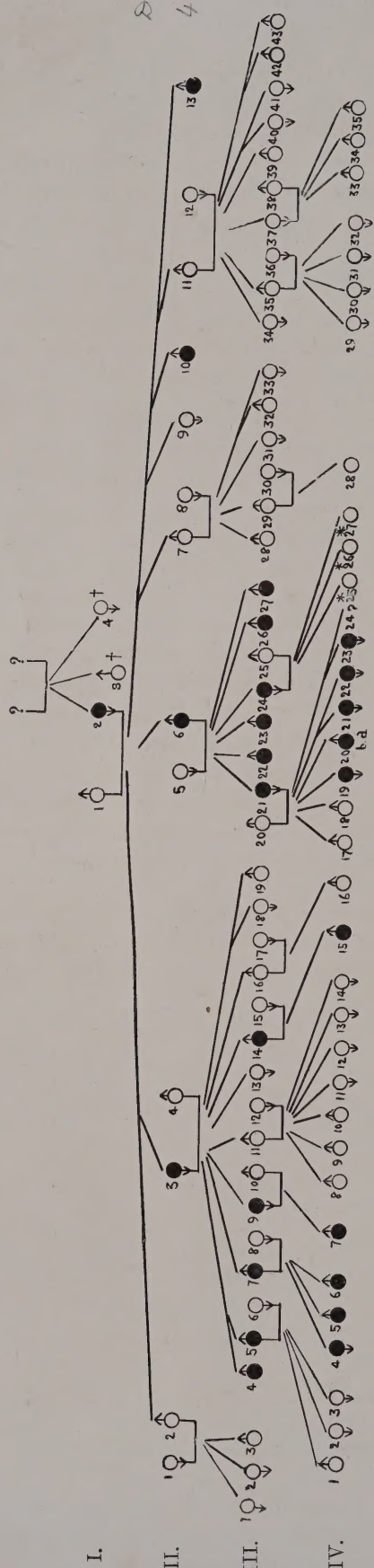
Lastly, I have most heartily to thank Mr J. H. Astbury, whose intimate knowledge of the district and its inhabitants has been most helpful to me in my inquiries.

(2) Before turning to more definite details as to the present family, I should like to mention the existence of a similar family, with names Bell and Agnew, at Whithorn, Wigtownshire, some 50 years ago. An old man told my informant that a boy with deformed hands was a descendant of the "Cleppie Bells." This was a family, one of which had assisted as Sheriff's officer at the drowning of the Wigton Bay martyrs in 1685. On that occasion the officer Bell said to a young maid, Margaret Wilson, "Will you not say: God bless King Charlie, and get this rope from off your neck?" "God bless King Charlie, if He will," she responded. Whereupon he said "Clep down among the partens and be drowned." Thus he was called "Cleppie Bell," and his descendants have ever afterwards suffered from a deformity of the hand, although sometimes a generation is missed over. It is true that the term "clepped" in Scotland refers to webbed, but the reference to the lobsters suggests "lobster-claw" deformity, and one phase of this, the syndactyly, might easily be described as fingers grown or webbed together. The boy referred to above became a sailor and was last seen in Glasgow 20 years ago. No stress whatever can be laid on such a tale as this, but it is interesting as pointing to the existence in Scotland* of a deformed hand inheritance for nearly two hundred years, and the information might be followed up by any one coming across Scottish cases of either webbed hands or lobster-claw. This is the only case I have met with in which there is any statement that deformed offspring were born from undeformed parents†. Throughout both my family and that of

* Messrs Lewis and Embleton state that their family originated in Scotland.

† This follows also from the somewhat different version given in Sir Andrew Agnew's *The Hereditary Sheriffs of Glasgow*, Edinburgh, 1893. We read in Vol. II. p. 142: "Still more grotesque is the tradition of the 'Cleppie Bells.' A constable who was held to have carried out his orders unfeelingly, as he fastened the women to the stakes, was asked how the poor creatures behaved when the cold wave roared and foamed about their heads. 'Oo,' he replied jocularly, 'they just clepped roun' the stobs like partens, and prayed.'—Soon after Bell's wife was brought to bed, when the howdie exclaimed in horror: 'The bairn is clepped!' (i.e. the fingers grew firmly together). Another child was born, and yet another, and as each little wretch in turn was seen to be 'clepped' the most incredulous were convinced it was a judgment of Providence.—We have been gravely assured that within the memory of man a female descendant of the bad constable on giving birth to a child, was horrified by the exclamation, 'The bairn is clepped.'"

FAMILY TREE. INHERITED "LOBSTER CLAW."



† No record of condition of I. 3 and I. 4.

* Died at birth, no record.

Messrs Lewis and Embleton undeformed members give rise only to undeformed offspring. It is not possible to say whether the deformity really is latent or not, there have been no cousin marriages to form even a rough test of latency.

The absence of cousin marriages of any kind compels us to consider "lobster-claw" as a dominant character, and after the ancestress I. 2 of my pedigree, who may in Mendelian terminology possibly have been a homozygote, all the descendants must be looked upon as heterozygotes. On this assumption all normal individuals are recessive, and the fact that normal individuals from tainted stocks do not have deformed offspring meets with a ready explanation.

It is this noteworthy point not only in the present family, but in Lewis and Embleton's family, in Drinkwater's Brachydactylous Family and in Nettleship's Night Blind Family, which is the main-stay in these cases of the Mendelian theory. It is perfectly true that it would flow from other hypotheses, for example from the assumption that the gamete was not pure, but that dominance flowed from a numerical preponderance of allogenic determinants*. Such a theory could only be tested by inbreeding. For the sake of science, therefore, if not for local well-being, it is desirable that marriages between the normal members of these stocks should occur, and this even in sufficient numbers to test whether there are really latent tendencies to the deformity in normal members of the stock. Should this not prove to be the case, the non-marriage of the deformed members would be sufficient to check the spread of the deformity. Obviously a determinantal theory not based upon the pure gamete would further account for the occasional and rare appearance of the deformity in a child of normal parents. Mendelism must assert in a case like the present where the character must be dominant, that such an appearance is a mutation or sport.

(3) Since the deformity must be looked upon as dominant in the Mendelian sense, it follows that on the average half the offspring of a deformed parent ought to be deformed. We have data for such families in three generations, and the following results flow from them:

N = Normal D = Deformed	Families					Totals
	1	*2*	*3*	*4*	*5*	
	N. D.	N. D.	N. D.	N. D.	N. D.	N. D.
II. Generation	4 4	—	—	—	—	4 4
III. Generation	5 5	0 6	—	—	—	5 11
IV. Generation	0 3	0 1	0 1	2 5	3 0	5 10
Totals	—	—	—	—	—	14 25

* See the paper in this number of *Biometrika* on p. 80.

The amount of material here is not very great, but, as far as it goes, it does not support the conclusion that for the tainted families the abnormal members are relative to the normal fewer in number. It seems rather to indicate that in the last generations the abnormal have been twice as numerous as the normal. My data are, however, far less numerous than Messrs Lewis and Embleton's. Leaving out their two doubtful cases, I find (*Biometrika*, Vol. VI. Plate I) that they have 32 normal to 43 abnormal members in the families of their stock with deformed parents. I shall confine my attention to their stock and my stock, because I feel in my own case the confidence of the personal collector, and am sure that a number of individuals have not been omitted or wrongly classed; and because I realise in their case also that Messrs Lewis and Embleton have examined their stock with the special view of testing definite ratios. The families of other observers may have been carefully worked out, but the importance of complete enumeration has only been recognised since it has become desirable to test Mendelian theory.

Examining the data from the statistical standpoint, I draw attention in the first place to the first family of the third generation. This family should approximate to 3 normal to 3 deformed, but every member born was deformed. The odds against a run of six deformed are 63 to 1, if the chances of normality and of lobster-claw are equal. The odds against a deviation as great as 25 to 14 from a ratio of equality are 14 to 1; the odds against Messrs Lewis and Embleton's ratio of 43 to 32 are about 9 to 1; the odds against such an excess as is represented by our combined results 68 to 46,—a ratio corresponding to 3 to 2 instead of 1 to 1,—are more than 49 to 1. Now this excess of the abnormal in any deformity like the present ought to be admitted, if it actually exists, for it is not only opposed to simple Mendelism, but its bearing on Eugenics is of the greatest importance. While I have found the like excess in other cases, it does not appear to be true for all deformities. Thus, from the pedigree plate in Mr Nettleship's admirable paper on Night Blindness*, I deduce the following results:

	Abnormal	Normal
Generation III.	10	6
Generation IV.	15	43
Generation V.	26	29
Generation VI.	30	51
Generation VII.	27	55
Generation VIII.	14	36
Generation IX.	9	11
Totals	131	231

* *Ophthalmological Society's Transactions*, Vol. XXVII. pp. 209–291.

The odds against such a deviation from equality are enormous, i.e. about 10,000,000 to 1. But it will be noted that the abnormal members are largely in *defect.* Mr Nettleship himself remarks that this deficiency may probably be explained by unavoidable imperfections in the record, especially in the earlier generations. The excess of normals, however, has been more than maintained in the recent generations; and if, in defending the Mendelian ratio of equality, we impugn the record on the ground that members of the stock are ashamed to own their defect and screen it, what then becomes of the evidence it undoubtedly provides for Mendelian theory in the "invariable continuity of descent of the disease in the affected branches, and its permanent disappearance from all other divisions of the genealogy"? The screening of the disease must clearly have become an hereditary character, and the above evidence of segregation would be worthless. This principle of segregation is so vital, is such a wonderful, and I am inclined to say, glorious addition to our knowledge of heredity, that I am inclined in these cases of family abnormalities to defend Mendel against Mendel, or to preserve Mendelian segregation at the expense of Mendelian ratios. We have seen that the odds against a ratio of 3 to 2, which is the combined result of our split-foot families, are very slight, and this applies equally to the case of Night Blindness, where a random sample deviating as much as the present one from the ratio of 3 to 2 would occur once on the average in 6 or 7 trials. There is an additional point also to be noted. The Curé of Vendémean, according to Mr Nettleship, states that if the original night-blind individual, "the Jean Nougaret of 1637 could reappear to-day, except for a few individuals recently established in the district, all could salute him as their ancestor" (*loc. cit.* p. 288). If this be true, then the Mendelian ratio to be expected is more than 1 to 1, for the husband or wife of the night-blind individual would, in certain cases, be screening their own defect, and the offspring ought to have been night-blind in the ratio of 3 to 1. On the whole, while these cases give very definite evidence of the segregation factor, they do not seem to me to favour the segregation in rigid Mendelian proportions.

(4) When we turn to the actual nature of the deformity in the case I am dealing with, we find all the principal types observed by Messrs Lewis and Embleton recur, with one exception which may exist, but owing to the few individuals who have been radiographed, this cannot be asserted. I refer to the existence of cross bones (*Biometrika,* Vol. VI. p. 41). On the other hand, individuals exist showing syndactyly and polydactyly; gross deformity, several "fingers" placed promiscuously on a large misshapen "hand"; or branching fingers which, without careful radiographic examination, it would perhaps be hard to classify as polydactyle or misplaced.

The usual type of hand is to outward appearance, the hook-like form of my Plate XI, but the degree of defectiveness in the bones of the hand, even as affecting the carpus, may vary widely. In one case (II. 3) the hands were perfect but the feet deformed. There seems to be some evidence that in the younger generations the defectiveness is increasing; thus all the children of II. 6 had two-

toed feet, but the children of III. 21 are one-toed, with one exception, who has one two-toed foot, and the other one-toed. In this family as distinct from that of Lewis and Embleton the defectiveness in the foot may reach the tarsus. Contractions of the fingers and toes, these being flexed towards the centre of the member, are common. The following is a rough external account of the deformity of the individuals in the pedigree:

List of deformed members of the Stock.

I. 2. Ann J. Nothing known of her parents; said to have had a brother and sister but no record of their condition to be found. She had one finger only to each hand, but the 1st and 5th toes on both feet. Died at 77. There was no consanguinity between I. 1 and I. 2.

II. 6. Two fingers on right hand, one finger and thumb on left; 1st and 5th toes on each foot. No consanguinity with wife II. 5. Cause of death, asthma.

II. 10, II. 13. Deformed, both hands and feet.

II. 3. Hands perfect, both feet only 1st and 5th toes. Husband II. 4 not related.

III. 21—24, 26, 27 are children of II. 6 above.

III. 21. One finger to each hand, and 1st and 5th toes. Subject to fits. Unrelated to husband, III. 20. See Plates XI, XII, XIII and XV, Fig. vii.

III. 22. Right hand six fingers, the supernumerary beyond the little finger; left hand five fingers, all deformed and bent at angles to palm of hand. 1st and 5th toes on each foot. Dead.

III. 23. Both hands one finger, both feet 1st and 5th toes. Diseased arm bone.

III. 24. Right hand ring and little fingers, but these united together; left hand two bent fingers and thumb. 1st and 5th toes on each foot.

III. 25. Right hand two fingers only; left hand two fingers and thumb; both feet 1st and 5th toes.

III. 26. One finger on each hand; 1st and 5th toes on each foot.

III. 4, 5, 7, 9 and 14 are children of II. 3. No details except that both hands and feet are affected.

In the fourth generation, we have first children of III. 21.

IV. 19. Both hands and feet affected; 5th toe only. Married, no offspring at present.

IV. 20. Both hands and feet affected; 5th toe only; stillborn.

IV. 21. Only 5th toe on each foot; little finger on each hand. Has had one or more fits. Plates XI, XIV and XV, Fig. viii.

IV. 22. Both hands and feet affected; 5th toe only; weakly constitution.

IV. 23. One finger on each hand. 1st and 5th toes on right foot, 5th toe only on left foot. See Plates XI and XVI.

IV. 24? Expected Birth*.

IV. 18 has also a weakly constitution though hands and feet are normal.

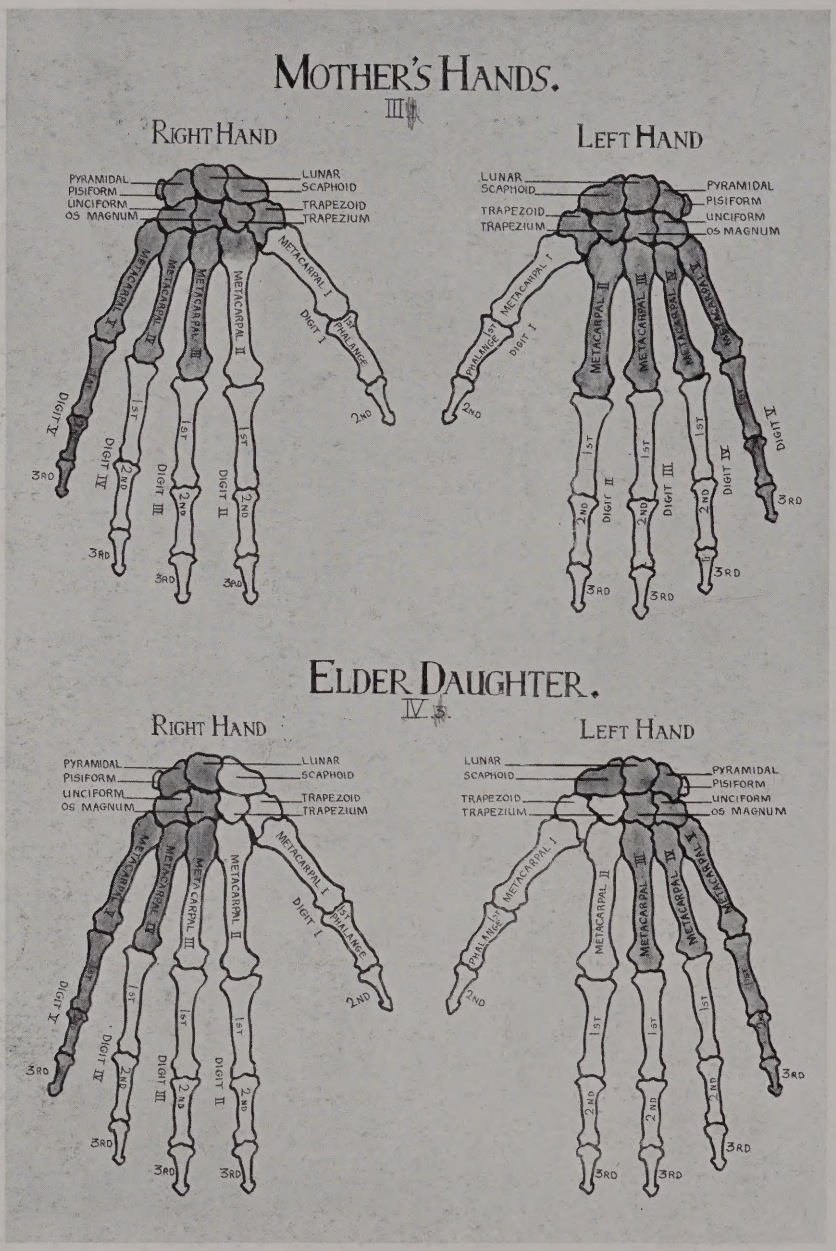
Of the descendants of II. 3, the grandchildren through III. 7 and III. 8 (who are unrelated) are:

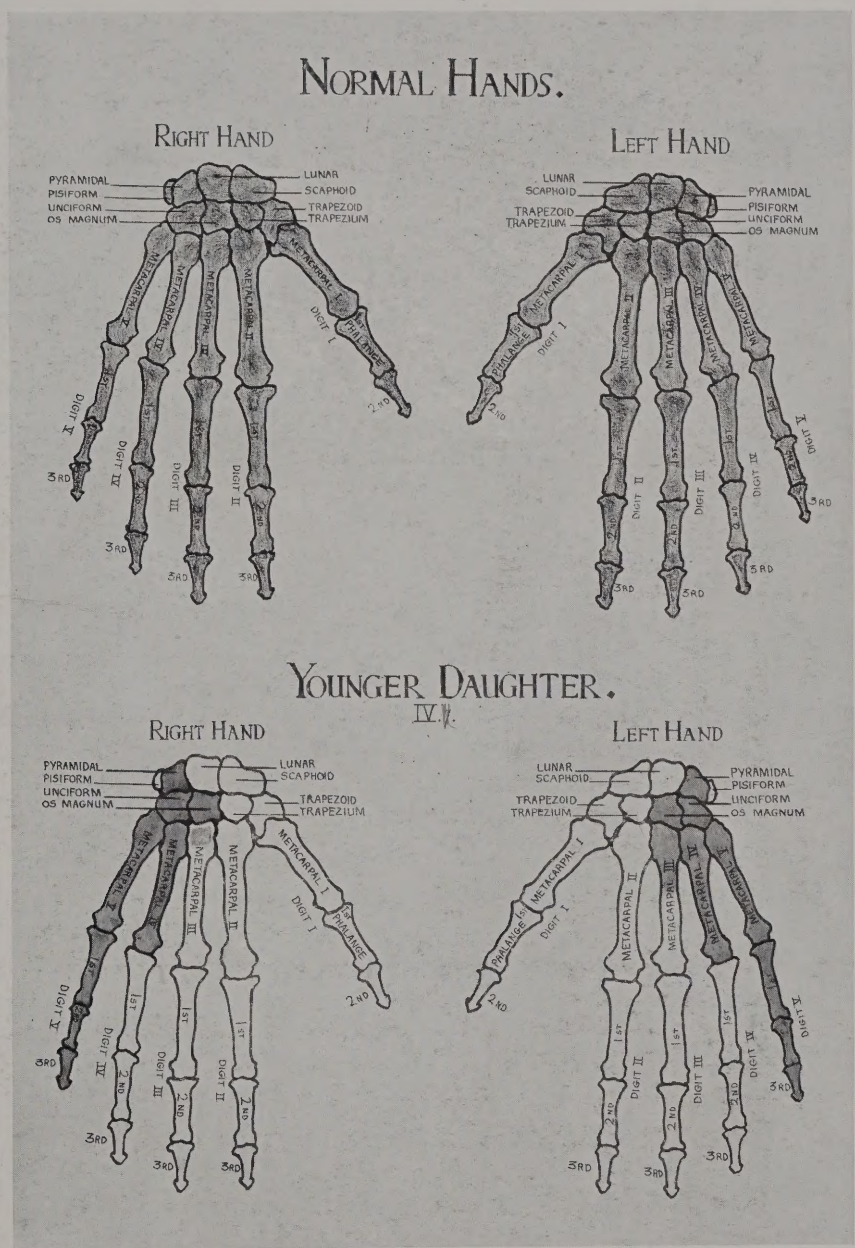
IV. 4. Ring and little finger only, joined together on each hand. 1st and 5th toes on each foot.

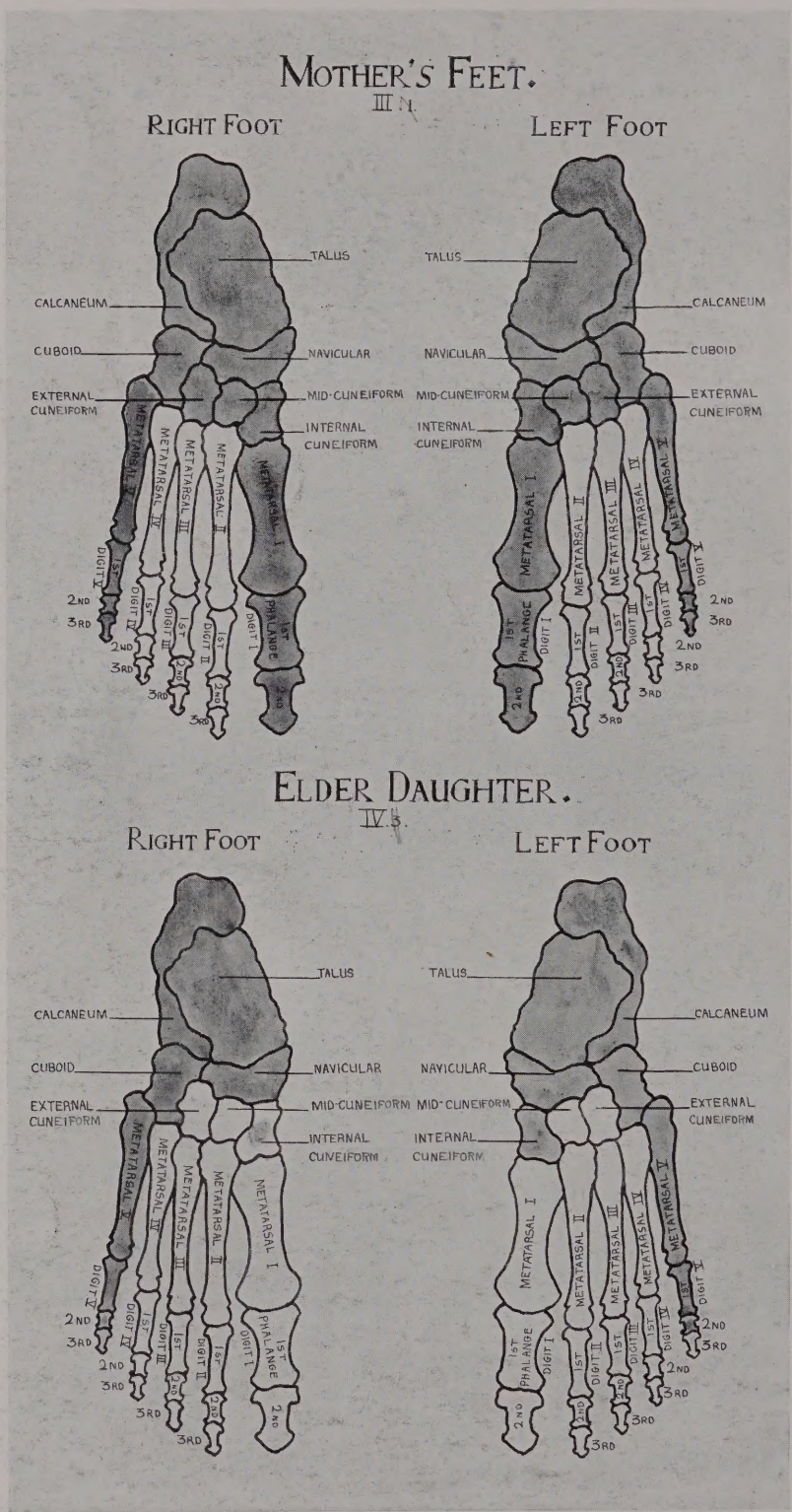
IV. 5. One little finger only on each hand; 5th toe only on each foot.

IV. 6. One little finger only on right hand; thumb and little finger on left hand; 1st and 5th toes on each foot.

* Born as this proof goes finally to press; a girl deformed in both hands and feet. Thus the improbability of the Mendelian quarter is greater than that given above.

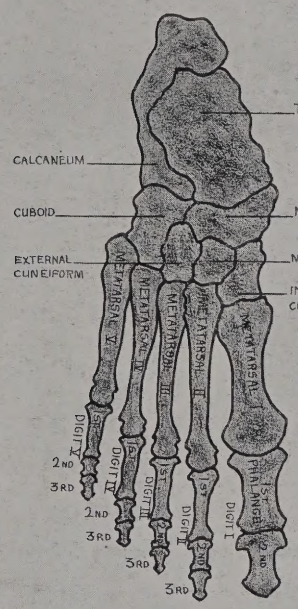

MOTHER'S HANDS.
III
RIGHT HAND
LEFT HAND
PYRAMIDAL
PISIFORM
UNCIFORM
OS MAGNUM
LUNAR
SCAPHOID
TRAPEZOID
TRAPEZIUM
METACARPAL I
METACARPAL II
METACARPAL III
METACARPAL IV
METACARPAL V
DIGIT I
DIGIT II
DIGIT III
DIGIT IV
DIGIT V
PHALANGE
1ST
2ND
3RD
ELDER DAUGHTER.
IV.
RIGHT HAND
LEFT HAND


NORMAL HANDS.
RIGHT HAND
LEFT HAND
PYRAMIDAL
PISIFORM
UNCIFORM
OS MAGNUM
LUNAR
SCAPHOID
TRAPEZOID
TRAPEZIUM
METACARPAL I
METACARPAL II
METACARPAL III
METACARPAL IV
METACARPAL V
PHALANGES
DIGIT I
DIGIT II
DIGIT III
DIGIT IV
DIGIT V
1ST
2ND
3RD
YOUNGER DAUGHTER.
IV.
RIGHT HAND
LEFT HAND

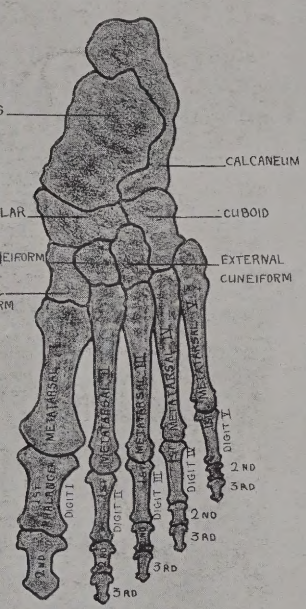

MOTHER'S FEET.
III.
RIGHT FOOT
LEFT FOOT
TALUS
CALCANEUM
CUBOID
NAVICULAR
EXTERNAL CUNEIFORM
MID-CUNEIFORM
INTERNAL CUNEIFORM
METATARSAL I
METATARSAL II
METATARSAL III
METATARSAL IV
METATARSAL V
1ST PHALANGE
DIGIT I
DIGIT II
DIGIT III
DIGIT IV
DIGIT V
1ST
2ND
3RD
ELDER DAUGHTER.
IV.
RIGHT FOOT
LEFT FOOT
TALUS
CALCANEUM
CUBOID
NAVICULAR
EXTERNAL CUNEIFORM
MID-CUNEIFORM
INTERNAL CUNEIFORM
METATARSAL I
METATARSAL II
METATARSAL III
METATARSAL IV
METATARSAL V
1ST PHALANGE
DIGIT I
DIGIT II
DIGIT III
DIGIT IV
DIGIT V
1ST
2ND
3RD

Normal Feet.

Right Foot



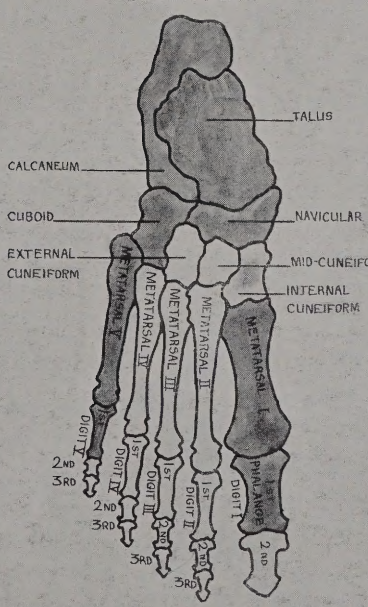
Left Foot



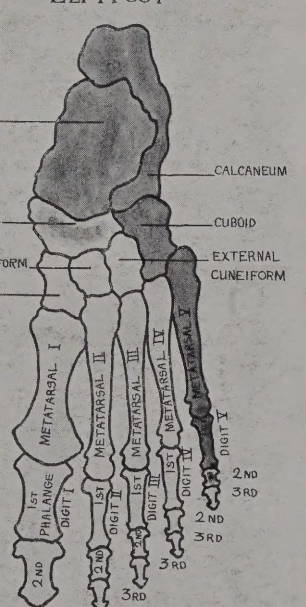
Younger Daughter.

IV. 7.

Right Foot



Left Foot



Of the other two grandchildren of II. 3, belonging to the deformed group, IV. 7 and IV. 15, I only know that both feet and hands are deformed.

Attention must be drawn to the fact that the deformed individual III. 5 has had three children all normal, and to the fact that the deformed individual III. 24 has had three children born dead, of whom nothing is known as to presence or absence of deformity.

(5) Of the three individuals III. 21 and her two daughters IV. 21 and IV. 23, aged respectively 10 and 2½ years, the following account has been drawn up by Professor Thane from Dr Mackenzie Davidson's radiograms. In the list below a plus sign marks the presence of the corresponding bone, the dot its absence. In the diagrams of Plates IX and X the defectiveness of the bones of the hands

Hands	Mother III. 21 Age 37		Elder Daughter IV. 21 Age 10		Younger Daughter IV. 23 Age 2½	
	Right	Left	Right	Left	Right	Left
Scaphoid ...	+	+	•	+	•	•
Lunar ...	+	+	+	+	•	•
Pyramidal ...	+	+	+	+	+	+
Pisiform ...	+	+	•	•	•	•
Trapezium ...	+?	+	•	•	•	•
Trapezoid ...	+	+	•	•	•	•
Magnum ...	+	+	+?	+	+	+
Unciform ...	+	+	+	+	+	+
Metacarpal 1	•	•	•	•	•	•
,, 2	+	+	•	•	•	•
,, 3	+	+	+	+	+?	+
,, 4	+	+	+	+	+	+
,, 5	+	+	+	+	+	+
Digit 1	•	•	•	•	•	•
,, 2	•	•	•	•	•	•
,, 3	•	•	•	•	•	•
,, 4	•	•	•	•	•	•
,, 5	+	+	+	+	+	+
	3 ph.	3 ph.	3 ph.	3 ph.	3 ph.	3 ph.

Feet	Mother III. 21		Elder Daughter IV. 21		Younger Daughter IV. 23	
	Right	Left	Right	Left	Right	Left
Calcaneum ...	+	+	+	+	+	+
Talus	+	+	+	+	+	+
Navicular ...	+	+	+	+	+	+
Intl. Cuneiform	+	+	+	+	+	•
Mid. Cuneiform	+	+?	•	•	•	•
Extl. Cuneiform	+	+	•	•	•	•
Cuboid ...	+	+	+	+	+	+
Metatarsal 1 ...	+	+	•	•	+	•
,, 2 ...	•	•	•	•	•	•
,, 3 ...	•	•	•	•	•	•
,, 4 ...	•	•	•	•	•	•
,, 5 ...	+	+	+	+	+	+
Digit 1 ...	+	+	•	•	+	•
	2 ph. fused	2 ph.			1 ph.	
,, 2 ...	•	•	•	•	•	•
,, 3 ...	•	•	•	•	•	•
,, 4 ...	•	•	•	•	•	•
,, 5 ...	+	+	+	+	+	+
	3 ph.	3 ph.	?2 ph.	2 ph	1 ph.	2 ph.?

and feet is indicated by noting the presence of bones by shading in each case, the unshaded bones are absent, a key figure indicates the bones of the normal hand and foot*. These figures show nothing of the contracted and distorted form of the original members, for which the reader is referred to Plates XII to XVI which give a few of the radiograms of these three cases. It is fairly possible by the shadow of the fleshy parts and the aid of Plate XI to reach a reasonable appreciation of the actual appearance of the living individuals.

The nature of the deformity in these limbs is a partial suppression of the digits, together with reduction (absence or fusion) of certain associated carpal or

* Owing to an oversight of the draughtsman, the names of the bones were inserted on *all* the figures; it seemed better to leave the redundancy than to go to the labour of redrawing the plates.

tarsal elements in the more pronounced cases. The bones of the forearm and leg are normal in their disposition as far as they appear in the photographs.

In the mother, III. 21, the left hand has a well-developed carpus which may be regarded as normal, while in the right hand the distal row of the carpus shows only three bones, the outermost of which probably represents the fused trapezoid and trapezium. In both hands the fifth digit (little finger) is well developed; the whole thumb is missing, as are also the phalanges of the index, middle and ring fingers, those digits being represented only by the metacarpal bones, of which the proper second (that of the index finger) is the smallest, and indeed in the right hand is reduced to a small piece of bone corresponding to the basal portion only of the normal bone.

In the left hand of the daughter, IV. 21, there are three bones in the proximal row of the carpus, the pisiform being as yet unossified, two only in the distal row, magnum and unciform, the three inner metacarpal bones, diminishing in size from the fifth to the third, and the normal three phalanges of the little finger. Suppressed are the two outer bones of the distal row of the carpus, trapezium and trapezoid, the first and second metacarpal bones, and the phalanges of the thumb, index, middle and ring fingers. The right hand shows a greater degree of reduction, the scaphoid being suppressed in the proximal row of the carpus, while in the distal row there is only one bone representing the fused magnum and unciform, and the third metacarpal is smaller than in the left hand.

IV. 23 has only three carpal bones, pyramidal, magnum and unciform, in each hand, but she is still too young for ossification to have started in the others; the fourth and fifth metacarpal bones are fairly well developed; the third metacarpal is small, in the right hand merely a nodular vestige of the basal portion; the phalanges of the little finger are present and of good size.

The malformation of the hands consists therefore of suppression or reduction of parts proceeding from the radial (thumb) side towards the ulnar (little finger) side, and carried to the greatest extent in the young child, IV. 23.

The feet are not so homogeneous as the hands, and fall into two well-marked groups. In the one, comprising the two feet of the mother and the right foot of the younger child, IV. 23, the three middle digits are suppressed, including metatarsal bones and phalanges, while the first and fifth are fairly well developed, especially in the mother. The tarsus may be regarded as complete in the mother, only in the right foot the number of elements is apparently reduced by the fusion of the internal and middle cuneiform bones; while in the right foot of IV. 23 the middle and external cuneiform bones are missing, but it may be that they are still in the cartilaginous stage, ossification not having yet begun.

The two feet of IV. 21 and the left foot of IV. 23, which constitute the second group, are also very much alike. Here the inner four digits are suppressed, and only the fifth (little) toe is represented. In all the talus, calcaneum and cuboid of the tarsus are well developed, the navicular is ossified (just beginning to ossify in

IV. 23)*, and the external and middle cuneiform are wanting, while a rudimentary internal cuneiform is present in both feet of IV. 21; that this is not seen in IV. 23 is again possibly due to delayed ossification.

Hence the second group differs from the first in the suppression also of the first digit, and probably a concomitant reduction of the tarsus. The close resemblance of the condition in this second group to that exhibited by the hands is obvious.

All through the deformity is greater in the children than in the mother.

In the hands, IV. 23, the younger daughter, shows greater degree of reduction than IV. 21. The small number of carpal bones in IV. 23 is to be explained however by the fact that the child is too young for ossification to have begun in the missing elements. The same consideration will account for the absence of the pisiform in IV. 21. This does not apply to the metacarpal bones.

In the feet there is a difference on the two sides. The left foot is slightly more reduced in IV. 23 than in IV. 21; whereas the right foot is much more defective in IV. 21, IV. 23 having the first digit fairly well developed.

In all three the right hand shows a greater degree of reduction than the left, expressed in the condition of the metacarpal bones.

In the mother the left foot may be regarded as being slightly more reduced, as indicated by the fusion of the internal and middle cuneiform bones. In IV. 21 the reduction is slightly greater in the right foot than in the left; whereas in IV. 23 the left foot is the most reduced of the whole series, while the right foot has a well developed metatarsal and phalanx to both great and little toes.

(6) I have delayed referring to my final criticism of the application of Mendelian conceptions to cases of abnormality like the present, until I had placed before the reader the above accurate account of the nature of the defect in two or three individuals. But having done so it seems to me that a very difficult question has to be answered. What in a case like this of split hand and foot is the Mendelian unit character? What are the allelomorphs whose combined absence or combined presence is impossible in the zygote? We can understand what is meant when an organ is said to be yellow or white, rough or smooth, however much we may wish for the use of a quantitative scale of such characters. We can understand what is meant by the presence or absence of a given individual bone, say the second phalanx of the fifth digit. But in a case like the present we are concerned not with a simple unit of any kind, but the absence of a complex of even as many as 60 bones situated in four different parts of the body. In other cases 10, 20 or 40 bones may be wanting or incompletely developed. It is true that these parts are highly correlated, but as we have seen, deformity of the feet is not always accompanied by deformity of the hands. Can we look upon the character here as built up of a series of coupled characters representing the system of bones we have referred to? If so, how can we explain the variety of detail which is to be found within the same family? How shall we account for other

* It is interesting to the anatomist to note that the tuberosity of the navicular bone has a distinct centre of ossification in both feet of IV. 21.

families in which mere syndactyly or polydactyly occurs, or again in which club-foot alone is inherited? It appears to me that in a case like the present we cannot possibly speak of the presence or absence of a unit character, there must be an inertia or weakening of some development-controlling determinant. I will not venture to call it an absence of such a determinant, because its range varies so widely from individual to individual. If we use the word absence we must make the full control depend on the presence of a large number of units, and there must be—as for example in the reducing division—some process by which a random number of these are cast out. Even with this interpretation we are really thrown back on quantitative degrees in the controlling factor. These control units cannot be specific, for if they were associated with special bones, it is not easy to understand how a bone occurring in parent and grandparent* could disappear in the zygote resulting from a normal union. The problem has been forced on me recently from the practical side in studying cases of human degeneracy with correlated defects, and although I have thought over it carefully I can see no daylight theoretically in these questions of correlated physical or mental defects —defects having a considerable range of variation, but peculiar to certain stocks —except from the assumption of development-controlling units or determinants.

These determinants may be more or less definitely associated with certain portions of the body—hands and feet, or eyes for example—but the total number rather than any specific character of each must determine in every individual case the extent of defective development. Whatever the *modus* may be, i.e. whether the determinantal system be quantitatively continuous or consist of discrete units, I venture to suggest that it is rather in the direction of inertness in controlling determinants than in the hypothesis of a definite allelomorphic character in the Mendelian sense that we must seek for light on the inheritance of physical or mental degeneracy or deformity in man. The complexity and variety of the deformity in many of these cases is too great for us to attribute it to a single allelomorph, and there is thus no obvious reason for anticipating a simple Mendelian ratio.

(7) On the subjects of my present study a few general remarks may be made. They show remarkable aptitude in the use of their misshapen hands. At school the children hold their own with normal scholars in writing, drawing, and even needlework. The family rather resent anything in the shape of pity. One of its men expressed himself as follows: "Bless 'e, sir, the kids don't mind it. They never had the use o' fingers and toes, and so they never misses 'em." One of the girls with the 5th digit only on each hand is learning dressmaking as a pursuit. In Messrs Lewis and Embleton's family the gait appears to be normal†, but I should hardly say that this describes those members of the present family that I have seen. One who has long been acquainted with them writes:

* E.g. the first digit of the foot existing in III. 21 and II. 2 disappears in the offspring of a marriage of III. 21 with a normal, as in IV. 21.

† *Biometrika*, Vol. VI. p. 28.

"Their feet lack the natural spring of the normal foot and their gait is therefore very ungainly, being assisted by a peculiar swing of the arms, much as a normal person would walk on heels alone. This is of course a great drawback, but not such as to incapacitate them from earning a living."

Turning to a further point we note that the deformed members seem to have no reduced fertility, nor do they appear to have difficulty in finding normal husbands or wives. On this account there is some concern at the perpetuation of the deformity in the district. Physically we may perhaps detect in some members signs of degeneracy, but they cannot be said as a rule to have weak constitutions. Intellectually the children take high rank in intelligence, or perhaps it would be better to say in "cuteness." Thus physically handicapped it would perhaps be unreasonable to expect in all members the highest social qualities, and assuming the stock to have been initially good, it may be doubted whether the deformity permits its members to select the physically and mentally fittest of normal mates. The stock is sufficiently extensive now to intermarry, and should it be ostracised might do so, a result which would have much scientific interest; but which might lead to the perpetuation of a split-foot race. Eugenically the problems associated with the family are not only scientifically difficult but practically serious; we live no longer in an age when the need for perfect hand and foot in the struggle for existence preserved the race from the perpetuation of a deformity. How is protection now to be maintained? In view of possible future legislation, it would seem for national purposes desirable that stocks of the present type should be kept under observation and careful records preserved of the history of all their branches. One of the most urgent social necessities is that every birth certificate should be associated with a medical certificate of the normality or abnormality of the child registered. A separate filing of the abnormal cases would not only keep the scientist in touch with important material, but provide the legislator with data whereby to appreciate the extent to which the suspension of natural selection is racially deleterious.

Hands of Mother (III. 21) and Daughters (IV. 21 and IV. 23)

Fig. i

Fig. ii

Feet of Mother (III. 21). Front view

Fig. iv

Fig. iii

Feet of Mother (III. 21). Side view

Fig. v

Fig. vi

Feet of elder Daughter (IV. 19). Front and side views

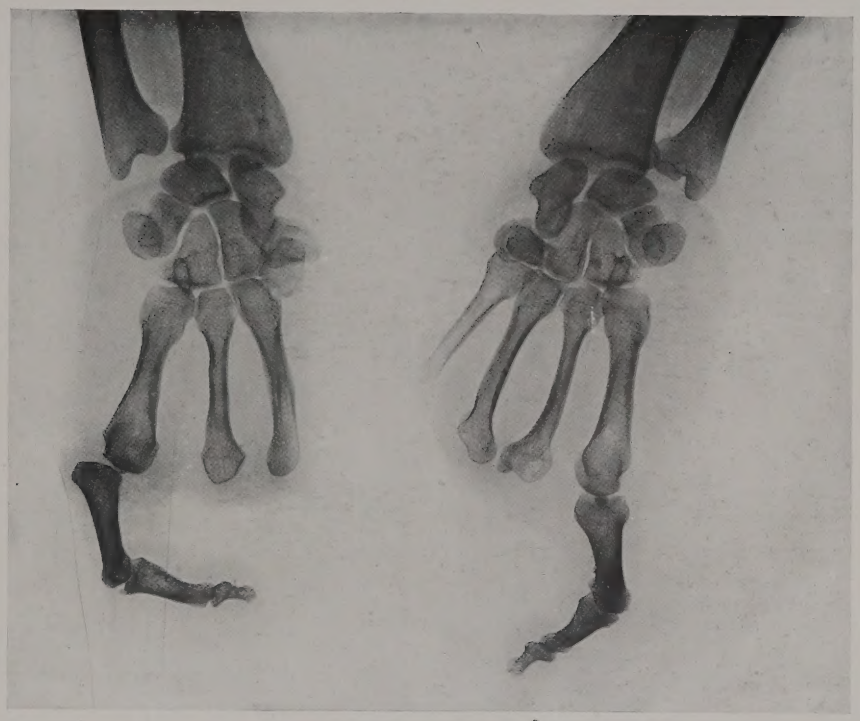


Fig. vii

Hands of Mother (III. 21)

Fig. viii

Hands of elder Daughter (IV. 19)

Fig. ix

Hands of younger Daughter (IV. 23)

Fig. x

Feet of younger Daughter (IV. 23)